

Degenerative lumbar spinal stenosis and lumbar spine configuration

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Abstract As life expectancy increases, degenerative lumbar spinal stenosis (DLSS) becomes a common health problem among the elderly. DLSS is usually caused by degenerative changes in bony and/or soft tissue elements. The poor correlation between radiological manifestations and the clinical picture emphasizes the fact that more studies are required to determine the natural course of this syndrome. Our aim was to reveal the association between lower lumbar spine configuration and DLSS. Two groups were studied: the first included 67 individuals with DLSS (mean age 66 ± 10) and the second 100 individuals (mean age 63.4 ± 13) without DLSS-related symptoms. Both groups underwent CT images (Philips Brilliance 64) and the following measurements were performed: a cross-section area of the dural sac, vertebral body dimensions (height, length and width), AP diameter of the bony spinal canal, lumbar lordosis and sacral slope angles. All measurements were taken at L3 to S1. Vertebral body lengths were significantly greater in the DLSS group at all levels

compared to the control, whereas anterior vertebral body heights (L3, L4, L5) and middle vertebral heights (L3, L5) were significantly smaller in the LSS group. Lumbar lordosis, sacral slope and bony spinal canal were significantly smaller in the DLSS compared to the control. We conclude that the size and shape of vertebral bodies and canals significantly differed between the study groups. A tentative model is suggested to explain the association between these characteristics and the development of degenerative spinal stenosis.

Keywords CT images · Degenerative lumbar spinal stenosis · Vertebral body dimensions · Lumbar lordosis · Sacral slope

Introduction

Neurogenic claudication due to lumbar spinal stenosis (LSS) is a frequent source of complaint of pain in the lower back and extremities, impaired walking and other forms of disability in the elderly [1–4]. Although the incidence of symptomatic LSS has as yet not been established, this condition is the most frequent indication for spinal surgery in individuals over 60 years [5, 6].

LSS can be developmental (sometimes called congenital) or acquired [7]. Developmental stenosis is rare, characterized by short pedicles and narrowing of the spinal canal dimensions [8, 9], whereas acquired degenerative stenosis is considered the most commonly observed type [10]. This type of stenosis arises in an advanced age and is essentially associated with degenerative changes of the three-joint complex [11–13].

Several studies have attempted morphometrically to characterize the lumbar spine in individuals with

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degenerative lumbar spinal stenosis (DLSS) with diverse results [3, 14–22]. Some radiological studies have reported that the changes in the anterior–posterior (AP) diameter, transverse diameter and cross-section area (CSA) of the spinal canal and dural sac are risk factors for developing spinal stenosis [16, 17, 23, 24]. Other studies focused on degenerative changes of the intervertebral disc, zygapophyseal joints and ligamentum flavum as dominant contributors of degenerative LSS [10–12, 25, 26]. Surprisingly, most studies neither scaled their measurements versus vertebral body size, nor considered the issue of lumbar spine alignment. Additionally, the poor correlation between radiological manifestations and the clinical picture [27–30] emphasizes the fact that more studies are required to determine the natural course of this syndrome.

Studying the morphometric characteristics of the lumbar vertebrae in the DLSS population may therefore assist in establishing a more comprehensive model for DLSS etiology and pathophysiology, and consequently in developing useful conservative treatments and improvement of surgical procedures. In addition, detecting specific features in an asymptomatic population can be important in preventing or delaying the onset of symptoms in this group.

The aim of this study was to find out the pathognomonic radiological features of the lower lumbar spine in symptomatic DLSS.

Materials and methods

Study groups

Two groups were studied. The DLSS group included 67 individuals (age 66 ± 10 years, sex ratio: 30 M/37 F) enrolled by a spine surgeon (KH) according to signs and symptoms related to LSS [31–33]. All individuals in this group exhibited intermittent claudication, often accompanied by other symptoms such as radiculopathy and/or LBP. In addition, all manifested a reduced cross-sectional area of the dural sac ($<100 \text{ mm}^2$) [23, 34, 35]. Individuals who suffered from developmental stenosis (AP diameters less than 12 mm) [36, 37], fractures, tumors, Paget's disease and iatrogenic (post-laminectomy, post-fusion) were excluded from this group. The control group was sex and age matched and included 100 individuals (age 63 ± 13 years, sex ratio: 51 M/49 F), referred for an abdominal CT scan. All these individuals were interviewed to exclude possible LSS-related symptoms.

CT measurements

All CT images were performed in the supine position with straight legs and obtained from the Department of

Radiology, Carmel Medical Center, Haifa, Israel. A multiplanar reformatting technique (Brilliance 64, Philips Medical System Cleveland Ohio) with a section thickness of 1–2 mm, MAS of 80–250 was used. All measurements were taken by one of the authors (JA) at L3 to S1 and divided into two main clusters.

Measurements in the axial plane

Anterior–posterior diameter of spinal canal (APDC) was measured at the mid-vertebral body at the basivertebral vein level (Fig. 1).

Cross-sectional area of dural sac (CSAD) was taken at the intervertebral disc level (Fig. 2).

Vertebral body dimensions, length (APBD) and width (WBD) were measured at the mid-vertebral body height (Fig. 3).

Measurements in the mid-sagittal plane

Lumbar lordosis (LL) was measured using a modification of Cobb's method [38] (adapted to the sagittal plane) (Fig. 4).

Sacral slope (SS) was measured using a modification of Ferguson's method [39] (adapted to CT images when the individual is in a supine position) (Fig. 5).

Lumbar lordosis and sacral slope measurements were in the supine position; nevertheless, it is well known that these measurements are similar to those obtained in a standing posture [40, 41].

Vertebral body height was measured at two points:

1. Anterior vertebral body height (AVBH) was measured as the distance between the most anterior superior

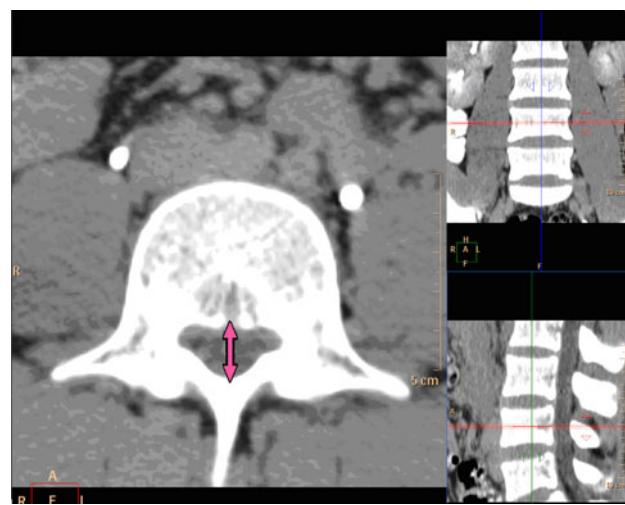


Fig. 1 Anterior posterior diameter of spinal canal

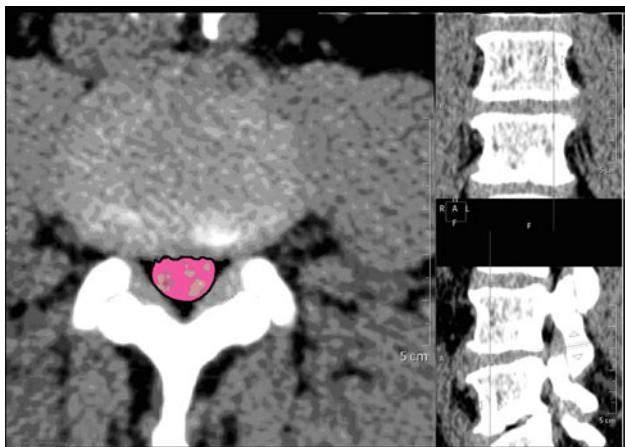


Fig. 2 Cross-sectional area of dural sac

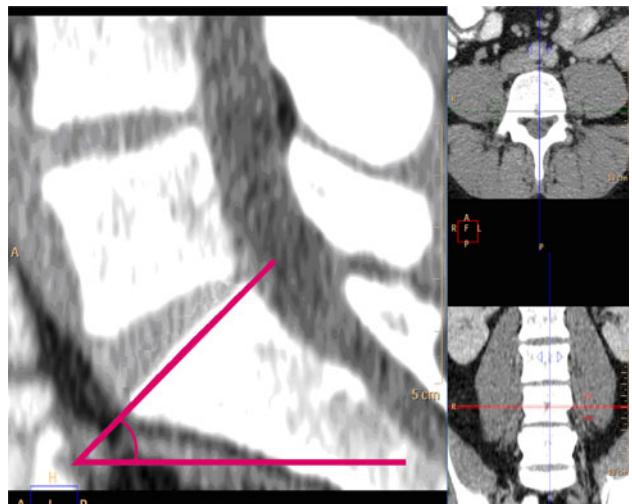


Fig. 5 Sacral slope measurement, following Ferguson's method

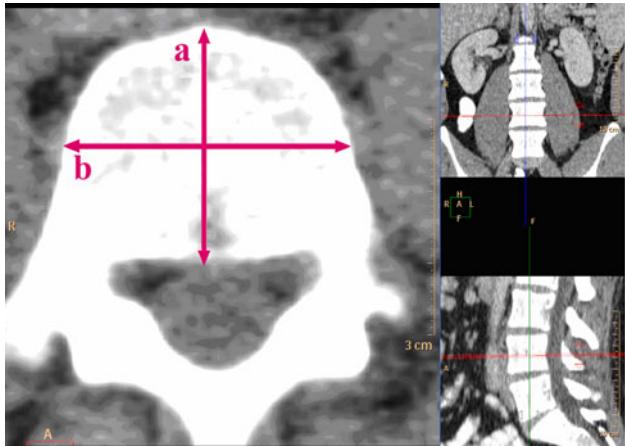


Fig. 3 Vertebral body dimensions; **a** vertebral body length and **b** vertebral body width

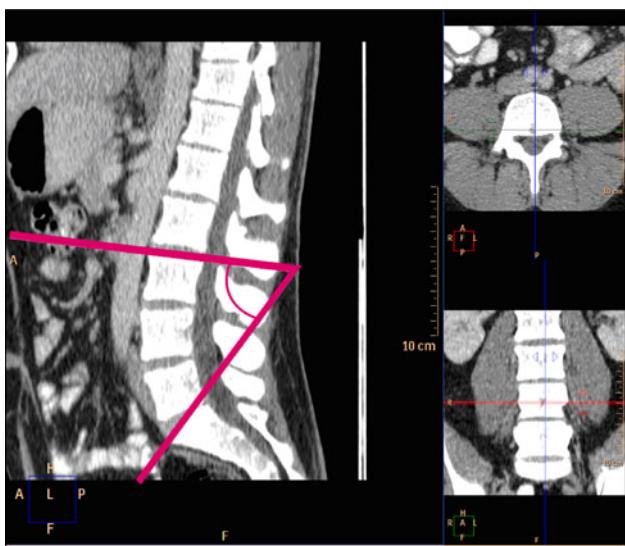


Fig. 4 Lumbar lordosis measurement, following Cobb's method

point to the most anterior inferior point on the vertebral body rim (Fig. 6, line a).

2. Mid-vertebral body height (MVBH) was measured as the distance between the two centers of the vertebral body surfaces (Fig. 6, line b). The degree of vertebral body beveling (VBB) was defined as the ratio of AVBH/MVBH.

Degenerative spondylolisthesis (DS) was also considered when the extent of listhesis was ≥ 2 mm.

Statistical analysis

All measurements were taken by one of the authors (JA). Fifteen individuals had repeated measurements (by JA and KH) carried out within 3–4 days after the initial measurements. The two testers were blinded to the result of the initial measurements. Intraclass correlations (ICCs) were calculated to gain insight into intra- and intertester

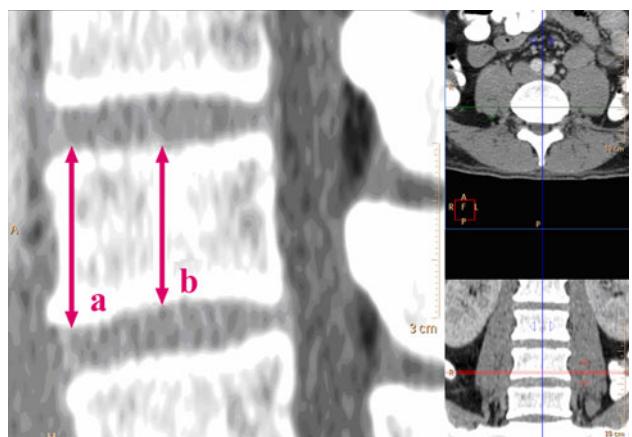


Fig. 6 Vertebral body height measurements: **a** anterior vertebral body height, **b** middle vertebral body height

reliability rates. A *t* test was carried out to determine the differences in the vertebral body and canal dimensions between the two groups. Chi-square test was performed to determine the association between lumbar stenosis and spine level. Significant difference was set at $P < 0.05$.

Results

Both intra- and intertester reliability rates were very high. The ICCs obtained for the intratester tests were: CSA = 0.930, APDC = 0.983, APBD = 0.984, WBD = 0.985, AVBH = 0.980, MVBH = 0.977, LL = 0.984, SS = 0.980 and DS = 0.933; and for the intertester tests: 0.890, 0.980, 0.982, 0.984, 0.974, 0.971, 0.981, 0.970 and 0.898, respectively. The prevalence of degenerative spondylolisthesis was significantly higher in the DLSS group compared to the control group (46 vs. 21%, $P = 0.001$).

The mean and SD values for all measurements are presented in Table 1.

AP diameter of the spinal canal

The DLSS group at all three lumbar levels studied had significantly smaller AP canal diameters compared to the control group ($P < 0.05$). Mean APDC was 15.3 mm at L3, 15.2 mm at L4 and 15.2 mm at L5, compared to 16.1, 16.7 and 17 mm, respectively, in the control group. Additionally, while in the control group the APDC values increased from L3 to L5; in the DLSS group they remained similar at all three levels.

CSA of dural sac

At all three lumbar levels studied, the cross-sectional areas of the dural sac in the DLSS group were smaller compared to the control ($P < 0.05$). The smallest mean area was at L4–5 (50 mm²) and the largest at L5–S1 (93 mm²). All mean values in the control group were greater than 120 mm².

Vertebral body dimensions

At all three lumbar levels studied, the APBD was significantly greater in the DLSS group compared to the control ($P < 0.05$). The mean APBD was 33.2 mm at L3, 34 mm at L4, and 34.1 mm at L5, compared to 31.7, 32.4 and 32.5 mm, respectively, in the control group. Similarly, vertebral body widths were greater in the DLSS group, yet significant only at L4 ($P < 0.05$). The mean VBW was 43 mm at L3, 45.2 mm at L4 and 49 mm at L5, compared to 42.4, 43.7 and 47.8 mm, respectively, in the control group.

Table 1 Measurements of the lower lumbar spine in the DLSS and control groups

Variable	DLSS Mean \pm SD	Control Mean \pm SD	<i>P</i> value
Spinal canal APD (mm)			
L3	15.3 \pm 1.8	16.1 \pm 1.9	0.006
L4	15.2 \pm 1.7	16.7 \pm 2	<0.001
L5	15.2 \pm 2	17 \pm 2.4	<0.001
CSA of dural sac (mm ²)			
L3–4	67 \pm 25	125.1 \pm 47	<0.001
L4–5	50.3 \pm 28	124.5 \pm 50	<0.001
L5–S1	93 \pm 38	137.6 \pm 55	<0.001
Vertebral body APD (mm)			
L3	33.2 \pm 3.2	31.7 \pm 3	0.005
L4	34 \pm 3.3	32.4 \pm 2.7	<0.001
L5	34.1 \pm 3.2	32.5 \pm 2.7	0.001
Vertebral body width (mm)			
L3	43 \pm 4	42.4 \pm 4	0.27
L4	45.2 \pm 3.7	43.7 \pm 3.9	0.005
L5	49 \pm 4.2	47.8 \pm 4	0.116
Anterior vertebral body height (mm)			
L3	27.1 \pm 2.7	29.7 \pm 2.5	<0.001
L4	27.9 \pm 2.6	29.6 \pm 2.1	0.001
L5	26.8 \pm 2.2	30.2 \pm 2.3	<0.001
Mid-vertebral body height (mm)			
L3	24.8 \pm 2.3	26 \pm 2.3	0.001
L4	25.4 \pm 2	25.9 \pm 2.1	0.124
L5	24 \pm 2.4	24.9 \pm 2.2	0.013
Vertebral body beveling			
L3	1.10 \pm 0.08	1.14 \pm 0.07	0.002
L4	1.10 \pm 0.08	1.15 \pm 0.07	0.001
L5	1.12 \pm 0.09	1.21 \pm 0.08	<0.001
Sacral slope (degree)			
L3	38.5 \pm 7	42 \pm 7	<0.001
Lumbar lordosis (degree)			
L3	42.8 \pm 7	45.2 \pm 7	<0.001

APD Anterior posterior diameter, CSA cross-sectional area

Vertebral body heights

AVBH were significantly smaller in the DLSS group at all three lumbar levels studied, compared to the control ($P < 0.05$). Mean AVBH was 27.1 mm at L3, 27.9 mm at L4 and 26.8 mm at L5, compared to 29.7, 29.6 and 30.2 mm, respectively, in the control group. MVBH was significantly smaller in the LSS group at L3 and L5, but not at L4. The mean values of MVBH were 24.8 mm at L3, 25.4 mm at L4 and 24 mm at L5, compared to 26, 25.9 and 24.9 mm, respectively, in the control group.

Vertebral body beveling

At all three lumbar levels studied, VBB was significantly smaller in the DLSS group compared to the control

($P < 0.05$). The mean beveling index was 1.1 at L3, 1.1 at L4 and 1.12 at L5, compared to 1.14, 1.15 and 1.21, respectively, in the control group.

Lumbar lordosis and sacral slope

Both angles were significantly smaller in the DLSS group compared to the control ($P < 0.05$). The mean lordotic angle was 42.8° in the DLSS group compared to 45.2° in the control; the sacral slope was 38.5° in the LSS group compared to 42° in the control group.

Location of stenosis

Stenosis usually presents as multileveled; however, it is rarely restricted to a single motion segment (Fig. 7). Stenosis at all three lumbar levels studied was the most common (47%), followed by stenosis at L3–4 and L4–5 (35%) and at L4–5 and L5–S1 (6.1%). Single level stenosis was present in 7.7% of the DLSS group at L4–5 and 3.1% at L3–4. No single case of stenosis was noted at L5–S1.

Discussion

Anatomically, LSS is caused by a reduction in the space available for neural elements as a result of changes in the osseous and/or soft tissue elements surrounding the spinal column [21, 42–46]. Several studies have suggested that DLSS begins with loss of disc height resulting in bulging of annulus fibrosis and foraminal stenosis. These changes alter the loading of the facet joints, leading to facet arthrosis, ligamentum flavum thickening and osteophytic overgrowth [10, 47]. It also affects the dural sac dimensions (CSA, AP) and often the interarticular diameters [35]. Typically, DLSS is often limited to a single level, particularly L4–5 [48–50], whereas in developmental stenosis narrowing of the canal is much more uniform. Verbiest [51, 52] introduced the concept of LSS in the early 1950s

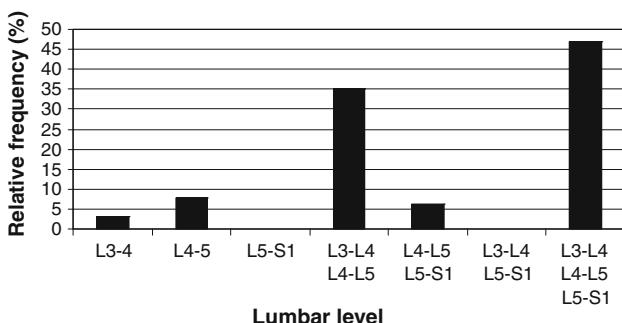


Fig. 7 Prevalence of stenosis in the lower lumbar areas, by motion segment

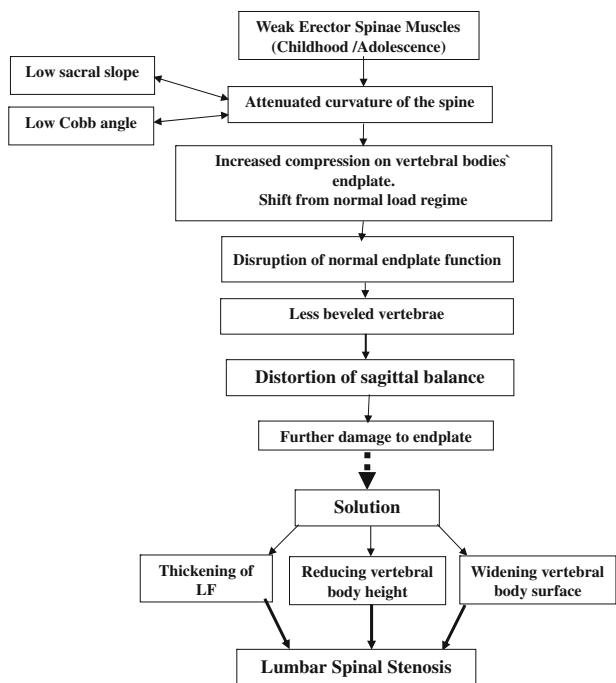


Fig. 8 Proposed model of DLSS development

and defined the absolute and relative AP diameter of spinal canal stenosis [31, 36, 37]. Later, Arnoldi et al. [7] classified LSS into subgroups, particularly degenerative, congenital and a combination of both. Eisenstein [16, 17, 53] measured the spinal canal in dried skeletons and suggested that mid-sagittal diameters of <13 mm should be considered stenotic. Studies based on CT scans of the lumbar spine confirmed Eisenstein's observation that the normal canal diameter is >12 mm [35, 54, 55]. Schonstrom et al. [35] found that in individuals who had undergone lumbar decompression, the AP diameter of the canal was 14.1 mm based on CT images. Contrary to the above, Postacchini et al. [56], based on skeletal data, cast doubt on the view that a mid-sagittal diameter of the vertebral canal of less than 12.0 mm was necessarily pathologic (the normal range varies among populations) and suggested that the diagnosis of developmental stenosis cannot be based solely on the dimensions of the spinal canal [56].

Albeit that A–P diameters in all three lumbar vertebrae were significantly smaller in the DLSS group compared to the control, the values obtained (15 mm) were still beyond the borderline for a "normal" A–P diameter. This is not surprising since in many cases the bony A–P diameter is normal even in the presence of severe constriction of the spinal canal [57]. This may imply that: (1) the borderline should be modified following a wide range population study [56] and (2) much of the lumbar stenosis may be due to the involvement of other structures (e.g., LF thickening) or degenerative changes (e.g., arthritis at the zygapophyseal joints) [57].

Several researchers have suggested that normal AP diameters increase as we descend from L3 to L5 [55, 58–61]. Interestingly, while the AP diameters of the bony canal in the control group follow this rule, in the DLSS it remains of the same magnitude in all three vertebrae. This emphasizes the fact that certain architectural bony features of the spinal canal may be involved in the development of degenerative stenosis.

The fact that almost half of the individuals in the symptomatic group in our study manifested degenerative spondylolisthesis lends support to the notion that DLSS commonly appears with degenerative listhesis [7, 62].

As in previous reports, CSAD with values of <100 mm² were considered the most sensitive parameter for manifesting clinical symptoms [23, 24, 34, 63, 64]. Moreover, the critical value was found at the level of L4–L5, which supported the notion that this segment was more susceptible to degenerative changes due to high compression and shear forces found at this level [65, 66].

The AP diameter of the vertebral body at L3, L4 and L5 and the vertebral body width of L4 were considerably greater in the DLSS group. Conversely, the vertebral body heights were significantly smaller at these levels (in both anterior and mid aspects). It is important to note that measurements for APBD and VBW were carried out at the mid-vertebral height to avoid the effect of endplate osteophyte formation. This implies that individuals with DLSS have wider and shorter lumbar vertebrae with minute wedging. It is noteworthy that since there are no available data demonstrating that these architectural characteristics predate the stenosis status, we cannot be absolutely certain that they are inevitably associated with the etiology of this phenomenon.

To the best of our knowledge, only scant information exists concerning vertebral body dimensions in DLSS. Zheng et al. [67] have revealed that the AP diameter of the spinal canal and the AP diameter of the vertebral body do not provide any evaluation in DLSS, contrary to the congenital type. Singh et al. [9] characterized congenital stenosis through MR imaging and stated that the AP diameter of the spinal canal, pedicle length and vertebral body width were significantly smaller in the stenosis group, whereas no difference was found in the vertebral body AP diameter and body height, compared to the control. Based on these studies, we concluded that there were no congenital components in our stenotic group.

Multilevel stenosis was more prevalent than a single level case. The stenosis at L3–4 and L4–5 was more common (35%) than at L4–5 and L5–S1 (6.1%). These findings challenge the notion that associates degenerative changes in the three-joint complex, mainly at L4–5 and L5–S1 [68, 69], with DLSS. According to this model, we would have expected DLSS to occur mainly at L4–5 and

L5–S1 and less at L3–4 and L4–5, contrary to our findings. We therefore believe that there must be other factors, in addition to the degenerative process, that most likely play a role in the development of DLSS.

There are three avenues one can take to explain the findings of this study. Certainly, they may be interrelated, yet still express different approaches in the quest for the best model to explain DLSS. The first and most straightforward one is to rely on genetics. This in itself does not offer an explanation for DLSS, but rather suggests that the unique shape of the vertebrae in DLSS patients has a genetic background. The second option focuses on the existence of primary disorder of the vertebral endplate. This approach offers a partial explanation for both the vertebral body and canal size deviation from the norms and raises some suggestions regarding the pathophysiology of DLSS. Following the studies of Epstein and Epstein (1990) and Lippit [71], it is possible that the disorder of the endplate could play a role in the development of abnormal dimensions of the vertebral body and spinal canal [70, 71]. Yet, we could not find any evidence in the English literature to support this hypothesis. The third avenue is to create a complex yet comprehensive model that combines all the data obtained in the current and previous studies on the spine of DLSS patients. Such a coherent model may offer an explanation of the origin and progress of this disease. Integration of all the morphometric characteristics of DLSS leads us to suggest the following scenario (Fig. 8): both lumbar lordosis and sacral slope start to appear when a child begins to acquire the upright position and subsequently increases until adulthood: age 10 years for sacral slope [72] and 17 years for lordosis [73]. Lumbar lordosis arises from the action of the erector spinae muscles (although other muscles are also involved). Because of the insertion of these muscles by a thick fascia onto the sacral spinous processes [74, 75], lordosis leads to horizontalization of the sacrum, i.e., verticalization of its endplate. A recent study has shown that the degree of lordosis is largely dictated by the balance between the trunk muscles, i.e., the weaker the extensor muscles of the back (relative to the flexor muscles) the smaller is the lordosis [76]. During childhood and adolescence, individuals with relatively weak erector spinae muscles may therefore fail to develop the necessary physiological lordosis for proper spinal balance and movements. The attenuated curvature of the spine will result in increasing load on the anterior aspect of the vertebral body endplate, the outcome of which will be a decrease in vertebral beveling. The rate of lumbar vertebral wedging depends on how much of the normal lumbar lordosis has been gained. When lordosis is poorly developed, there is less pressure on the posterior part of the vertebral body. Following Hueter–Volkmann's law, increasing pressure on the endplate of immature bone

retards growth and conversely reduced pressure accelerates growth [77] and thus the outcome is less beveled vertebrae. Therefore, the vertebral body anteriorly and LF posteriorly must take a major role in balancing the lower lumbar spine and handling the increased spinal load in these people (due to changes in the position of the line of gravity). The outcome is thickening of LF [78–83] and modifications of vertebral bodies, i.e., increasing vertebral body surface and decreasing vertebral height. All these alterations may lead in time to degenerative changes of the osseous and non-osseous tissues around the spinal column causing stenosis. The exact mechanism underlying the unique morphometric characteristics of the vertebral bodies in DLSS patients is yet not clear. One likely agent is damaged endplates: the presumed increased imbalance of the trunk muscles in our patients due to relatively weaker erector spinae muscles (evident by poor lordosis) and changes in spinal stress regime could alter endplate integrity. The endplate is the weakest link in compression during the vertebral body growth period and therefore can be damaged by accumulation of force on its posterior part as is the case in DLSS patients. This situation may lead, in time, to fatigue failure, followed by growth disturbances of the vertebral bodies, mainly of the posterior parts. The adaptive remodeling may result in a broader and longer vertebrae body and smaller spinal canal, as the posterior parts of the endplate are mostly involved.

The importance of the trunk muscles' strength, on the one hand, and proper lordosis, on the other, to the stability and proper posture of the spine is exemplified via the many syndromes that involve significant muscles' weakness (e.g., Duchenne muscular dystrophy). In many of these cases, the individuals adapt a compensatory lordotic posture to maintain spinal stability (via the locking mechanism of the posterior articular joints) and delay postural malformation [84].

Limitation of the study

Our hypothesis is partially based on Hueter–Volkmann's law, which is a general law and can be interpreted in various ways depending on the magnitude of the load (which we do not know).

Clinical implication

When segmental instability or severe discopathy is present in patients with DLSS, knowing the actual size and shape of the lumbar vertebrae may assist the surgeon in choosing adequate interbody devices and the most appropriate screws and plates to fix the different lumbar segments, following lumbar decompression surgery.

Conclusions

The size and shape of the vertebral bodies and vertebral canal significantly differed between the DLSS and the control. A tentative model is suggested to explain vertebral modification in the DLSS group. We believe that further research is required for deciphering the etiology and pathophysiology of DLSS and determining the association between erector spinae muscles strength and degenerative LSS.

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